# EXHIBIT L

**UNITED STATES DISTRICT COURT**
**SOUTHERN DISTRICT OF WEST VIRGINIA**
**AT CHARLESTON**

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| **IN RE: ETHICON, INC. PELVIC REPAIR SYSTEM PRODUCTS LIABILITY LITIGATION**<br><br>**THIS DOCUMENT RELATES TO:**<br><br>***Wave 1 Cases*** | **Master File No. 2:12-MD-02327**<br>**MDL No. 2327**<br><br>**JOSEPH R. GOODWIN**<br>**U.S. DISTRICT JUDGE** |

# Expert Report of Hannes Vogel, MD

## Qualifications

I am a physician board certified in anatomic pathology, neuropathology, and pediatrics. I am currently Professor of Pathology and Director of Neuropathology at the Stanford University School of Medicine. I am licensed to practice medicine in the State of California. I received my medical degree from Baylor College of Medicine, Houston, Texas, in 1980. My post-graduate training included residency training in pediatrics at Baylor Affiliated Hospitals and the University of California, San Francisco, where I served as chief resident in pediatrics from 1983-84. I completed residency training in anatomic pathology at Beth Israel Hospital/Harvard Medical School in 1987. I completed residency training in neuropathology at Stanford University Hospital/Stanford Medical School in 1989. I also completed a residency in pediatric pathology at Texas Children's Hospital in Houston, Texas in 2001. I am a member of the United States and Canadian Academy of Pathology, the American Association of Neuropathologists, and the German Association of Neuropathology and Neuroanatomy.

I have received numerous grants to perform research in the field of neuropathology, and I have taught neuropathology at Stanford University School of Medicine since 2002. I am a co-author of over 170 peer-reviewed articles published in medical journals, I serve on the editorial board of Clinical Neuropathology, and I am an ad hoc reviewer for several scientific and medical journals. I am the author of Nervous System (Cambridge Illustrated Surgical Pathology Series, Cambridge University Press, 2009). I regularly lecture to medical students, residents, and other physicians on a variety of topics in neuropathology. A significant portion of my work in neuropathology is focused on the diagnostic pathology of peripheral nerves, involving diagnostic nerve biopsies and questions regarding peripheral nerve tumors as well as traumatic neuromas. I am routinely consulted by diverse members of the pathology faculty at Stanford, including gynecologic pathologists and genitourinary pathologists, on questions of neuropathology involving pelvic tissues. In addition, I am consulted on essentially a daily basis regarding neuropathology issues by pathologists and other physicians from throughout North America, Mexico and Central America, and Europe. My particular expertise in nerve and muscle pathology is reflected in the volume of consultative material I receive pertaining to nerve and muscle biopsies, and as a faculty member of the Stanford Neuromuscular Service overseen by the Department of Neurology, in which I hold an appointment.

I held a position of responsibility for writing test questions in neuropathology, including nerve and muscle pathology, on the American Association of Neurologists Committee for preparing the residency inservice training examination used in every accredited neurology-training program in the United States between 2005 and 2012. I have recently provided test questions in the field of nerve and muscle pathology for the analogous inservice examination of

neuropathology fellows in the U.S., administered by the American Association of Neuropathologists.

As Director of Neuropathology, I am ultimately responsible for the timely and accurate issuance of diagnoses involving neurosurgical and autopsy tissues, promote research, and engage in teaching all aspects of neuropathology to neuropathology fellows in the fully accredited Stanford Neuropathology Fellowship, as well as residents in pathology, neurology, neurosurgery, rheumatology, endocrinology, and Stanford medical students. A more detailed statement of my professional background and experience with a list of my publications and my presentations to professional societies is contained in my curriculum vitae attached hereto as Exhibit A. A list of materials I have reviewed for these cases is attached hereto as Exhibit B. I anticipate reviewing the deposition transcripts of certain of plaintiff's experts in these cases and may further develop my opinions after having had that opportunity.

I have been asked to review medical literature, written opinions of Dr. Vladimir Iakovlev, and Dr. Iakovlev's testimony in the matter of allegations pertaining to possible consequences of the placement of mid-urethral mesh slings used to treat stress urinary incontinence, and specifically, Ethicon's meshes used to treat pelvic organ prolapse and those for stress urinary incontinence. I hold the opinions that follow to a reasonable degree of medical and scientific certainty and/or probability.

**Neuropathology**

Neuropathology, a subspecialty of anatomic pathology, and closely related to neurosurgery and neurology, is the study of diseases of the brain, spinal cord, their surrounding tissues, and muscles and nerves. Neuropathologists are extensively trained to examine the tissues

of the nervous system, through biopsies or autopsy excision of tissues, for study and diagnosis of diseases that involve the nervous system or its related tissues.

The human nervous system is easily the most complex system in the body but is most simply divided into the central and peripheral systems. The peripheral nervous system relays and delivers information from outside the central nervous system via a highly complicated system of nerves originating in the spinal cord and brainstem. Within the peripheral nervous system are the autonomic and somatic systems. The somatic system is composed of motor and sensory nerves. Motor nerve fibers are responsible for producing muscle contraction in skeletal and smooth muscle innervated by those fibers, and sensory fibers convey sensations of body position, temperature, light touch and deep pressure and pain. This network of nerves is composed of large nerves that then divide into increasingly smaller branches as they ultimately terminate in their targets such as muscle and various sensory receptors. Sensory fibers receive the sensations they convey through receptors tied to specific sensations. Another critical portion of the peripheral nervous system is the autonomic nervous system, which governs a wide variety of unconscious but life-sustaining actions, such as maintaining blood pressure, the beating heart, digestion, and others.

The term “nerve twig” is applied to the smallest branches of the peripheral nervous system, analogous to twigs of a tree branch. The vast majority of the larger peripheral nerves contain a mixture of motor, sensory and autonomic fibers, and there is no way to simply assign the functions to a nerve by their microscopic appearance. Most pathologists can recognize nerves in surgically excised tissues, but neuropathologists are best equipped to address issues of optimal processing of tissues for the diagnosis of nerve diseases, and recognition of true pathology in peripheral nerve tissues. Furthermore, there is no precedent for the diagnosis of intrinsic diseases

of peripheral nerves by examining nerve twigs that are found incidentally in surgically excised tissues, regardless of the presence of other microscopic findings in such tissues. For example, it is a doctrine of muscle pathology that nerve pathology cannot be diagnosed in the nerve branches and twigs that are found incidentally in all muscle biopsies. Authorities in the field state that the chosen nerve for biopsy interpretation should be accessible to conduction studies and have a predictable anatomical course and fascicular arrangement (Bilbao and Schmidt, in "Biopsy Diagnosis of Peripheral Neuropathy, 2015, p. 8).

The light microscope is an important tool in neuropathology, as it is for all anatomic pathology, but it is not without its limits. The analysis of nerves under the light microscope is limited in several respects relevant to the questions presented in these cases. For example, the microscopic appearance of all tissues that undergo obligatory fixation and chemical processing in preparation of slides to view under the microscope, especially the peripheral nervous system components, is susceptible to artifacts that are widely known to neuropathologists and general surgical pathologists alike. Fixation is known to cause shrinkage, which can lead to deformation or twisting of nerve fascicles and can cause nerves to lose their natural appearance in the body. (Bilbao and Schmidt, Chapter 7 Examination of the peripheral nerve biopsy. in "Biopsy Diagnosis of Peripheral Neuropathy, 2015, p. 123-126). Thus, conclusions about the "abnormal" shape of nerves or nerve twigs can be of questionable validity. Formalin fixation and paraffin embedding also creates artificial spaces that appear as small slit-like spaces in tissues. (Journal of Cancer 2015 6:759 -766; "The Muscle Biopsy" in: Myology, 3rd Ed. 2004, p 683). Thus, the assertion that such spaces may represent edema cannot be made with certainty, and therefore cannot lead to the conclusion that such spaces represent a disease process.

The body's nerves are diverse and are responsible for many distinct functions. The types of fibers in peripheral nerves differ in size and structure. Peripheral nerves are composed of nerves either of the somatic (*i.e.*, voluntary) nervous system or the autonomic (*i.e.*, involuntary) nervous system. Pathologists – even those trained and experienced in neuropathology such as myself – are not able to examine a histology section under the light microscope and determine whether nerve twigs in the field are sensory, motor, or autonomic in nature. Stains such as immunohistochemistry for S100 and neurofilament, which can aid in the identification of parts of nerves – specifically Schwann cells and axons, respectively – are incapable of differentiating between sensory, motor, and autonomic nerves. Moreover, S100 and neurofilament do not identify sensory receptors.

Thus, peripheral nerves are often mixed and mediate both motor and sensory function, but the extent to which a peripheral nerve transmits pain cannot be determined by its microscopic appearance. Even if a nerve fiber is sensory, the type of sensation it carries cannot be determined without identification of the sensory receptor that creates the signal or an understanding of where the nerve projections synapse in the spinal cord.

By contrast, the absence of nerve fibers may be associated with loss of sensation or other peripheral nerve function, but this is a difficult determination to make without considerable expertise as well as dedicated processing of tissues for neuropathological examination.
It is a normal histological finding to see nerve branches in all manner of surgically removed tissues, and there is no precedent in surgical pathology or neuropathology for assigning painful sensations that may have been reported by a patient preoperatively to the presence of such nerve fibers in explanted tissues.

**Innervation of the Pelvic Floor**

The female pelvic floor receives its nerve supply through a complex network of mixed peripheral nerves as described above. (Netter FH. "Innervation of reproductive organs". In: Nervous System. The Ciba Collection of Medical Illustrations. 1983, pp 88-89). The majority of the innervation is autonomic. Anatomically, the nerves supplying the region are connected to the spinal cord through lower spine openings or foramina. The nerves form networks or plexuses and the main plexus serving the pelvic floor is the hypogastric plexus. The autonomic nerves supplying the female genital organs include a superior portion serving the ovaries and outer uterus, a middle group also supplying the ovaries and uterus, and an inferior group supplying the lower uterus and cervix. There are tiny ganglia around the upper part of the vagina that help control the muscle and mucous coats of the vagina and urethra. Many of the sensory nerves in the pelvic region are distributed in the perineal and external genitalia. The urethra is innervated by the vesicular (bladder) plexus and pudendal nerve.

**Response to Plaintiff's Expert**

The plaintiff's surgical pathologist, Dr. Iakovlev, has proposed sweeping theories regarding nerve involvement and pain in the use of mid-urethral slings. Dr. Iakovlev's observations and theories regarding nerves are misplaced and without any reliable foundation in the field of pathology. Similarly, his attribution of clinical symptoms to his histology findings is completely without reliable basis in the field of pathology. Dr. Iakovlev does not possess subspecialty certification or expertise in neuropathology. Dr. Iakovlev's use and citation of medical literature – both in his report and in his recent publications – is often inappropriate because, as shown below, it is highly selective, skewed, and out-of-context such that it does not provide any actual support for his conjectures. His histological examination also shows

occasional nerve twigs adjacent to mesh material that he asserts represent "ingrown nerves", but which are of unproven clinical or pathological consequence.

Surgical interruption of the microscopic nerve supply to all manner of soft tissues in the human anatomy, whether in the presence of artificial materials or not, is always accompanied by fibrosis and reparative growth of blood vessels, nerve fibers, and other vital connective tissues. This is a necessary process, and typically occurs without painful sensations. Dr. Iakovlev's own recent review found low rates of pain in patients implanted with retropubic slings. (Safety considerations for synthetic sling surgery. Nat Rev Urol. 2015;12:481-509). A recent Cochrane analysis found comparable rates of dyspareunia in pelvic organ prolapse repair using polypropylene mesh versus the rates for native tissue repair (Transvaginal mesh or grafts compared with native tissue repair for vaginal prolapse (Review). Cochrane Collaboration 2016; pp. 5, 8). Furthermore, Dr. Iakovlev co-authored a publication in 2014 that showed a statistically similar number of nerve fibers in tissues removed after hernia repair without mesh as compared with tissues associated with removed mesh implants (Int. J. Clin. Med. 5, 799–810 (2014).

Dr. Iakovlev has also published the results of histological analysis of explanted mesh used in hernia repair in which the objective was to compare nerve densities in explanted polypropylene meshes in patients with or without chronic pain. The study is questionable in several ways. First, the clinical information is incomplete, with no mention of either the incidence of pain prior to mesh implantation in the two cohorts or whether pain resolved after mesh removal for pain. Second, nerve densities were, for undisclosed reasons, limited to nerves within the actual mesh plate and, by the authors' statistical analysis, more numerous in patients with removal of meshes with pain than in those with mesh removal for hernia recurrence.

Third, the study includes nonsensical remarks such as, "As shown in Fig. 1a, b, the caliber of nerves ingrown into the mesh pores ranged from nerve twigs to sizeable nerves up to 1 mm in thickness." The assertion that nerves of 1 mm diameter represent an ingrowth process is erroneous, because regenerating nerves in a surgical bed grow by a nerve-sprouting process and not as 1-mm fascicles.

Although a single patient with pain and recurrence had a markedly increased number of mesh nerve fibers, the significant overlap in nerve densities within mesh between the recurrence and pain group indicates that, aside from a minor statistical difference, patients from either group have similar nerve fiber densities in mesh. As is the case with his transvaginal mesh analyses, Dr. Iakovlev has not established anything that would enable other pathologists to predict which patients would experience pain based on nerve-fiber densities.

**Response to Dr. Iakovlev's Published Medical Literature**

Dr. Iakovlev has co-authored a recent article dealing with a variety of issues related to synthetic sling surgery using mesh implants. (Safety considerations for synthetic sling surgery. Nat Rev Urol. 2015;12:481-509).

Dr. Iakovlev's review article describes an incidence of chronic pain, defined as pain lasting greater than 6 weeks, in approximately 2.0% of patients undergoing mesh implantation with a retropubic sling. He cites a figure of 4.3% in overall patients "requiring" surgery who report pain, not otherwise specified, but notes that the symptoms leading to surgery are not mutually exclusive. For this reason, it is not possible to determine if pain as a solitary symptom was sufficient to lead to surgery. Importantly, Dr. Iakovlev's review article does not link any pathological findings with a one-on-one specific complaint in any given patient.

Dr. Iakovlev states that "[t]wo different kinds of pain caused by nerve injury have been suggested: centrally mediated hyperalgesia and a peripherally mediated painful hypoalgesia, suggesting the need for mechanism-based classification of neuropathic pain." (Pain 96, 141–151 (2002). Yet, the reference he cites does not involve patients who had undergone surgery or reported pelvic pain. Any applicability of centrally mediated hyperalgesia (meaning those with injuries to the central nervous system) to mesh surgery is tenuous. The remaining category – "peripherally mediated painful hypoalgesia" – for the painful syndromes described by Dr. Iakovlev appears to be incongruous with his theory that implanted meshes result in an *increase* in pain sensation, because hypoalgesia refers to *decreased* sensitivity to painful stimuli.

Dr. Iakovlev's review article then states that "few of the case studies of patients with SMUS-related pain quantified the severity of this pain, its character or how the pain affected patients' quality of life." Both the references Dr. Iakovlev quotes and the highly speculative nature of his assertions are equally deficient in proving his hypothesis. Furthermore, a recent study that examined the relationship between pathology and pain in patients with explanted synthetic midurethral slings is *not* quoted. Specifically, this deficiency in Dr. Iakovlev's work is shown by the findings of Hill and colleagues in "Histopathology of excised midurethral sling mesh." (Int Urogynecol J. 2015, 26:591-5). The Hill study looked at 130 explanted meshes and compared the histology of patients who complained of pain and those who did not. Contrary to their own hypothesis, these authors did not find increased inflammation in patients with pain. The authors also found no difference in fibrosis between the two groups. Dr. Iakovlev did not cite this article in his Nature Reviews Urology review and it is not found on the reliance list for his expert report. Dr. Iakovlev's recent testimony in another TVT case indicates that he simply did not locate the Hill paper, which was published electronically in November 2014. (Sept. 11,

2015 Deposition of Dr. Iakovlev, p. 145). Dr. Iakovlev has apparently acknowledged the Hill publication, but does not address it in his report. (Nov. 5, 2015 Deposition of Dr. Iakovlev, pp. 60-61)

Dr. Iakovlev's review article also states, "Pain in patients with a SMUS has been attributed to direct nerve injury during implantation, nerve entrapment, haematoma, infection, chronic inflammation, structural changes to the implanted mesh (shrinkage, stiffening, hardening and/or banding) and scarring. 38,330". These sweeping attributions are not directly supported by references 38 (Am. J. Obstet. Gynecol. 202, 481.e1–e5 2010) or 330 (Obstet. Gynecol. 119, 428–431, 2012). Indeed, reference 38 describes dyspareunia as a complication, and reference 330 is a single case report describing immediate postoperative pain which persisted until successfully managed by a nerve block. Interestingly, reference 38 makes the following statement in the Introduction: "polypropylene mesh was used to provide the midurethral support with no tension. Initial reported cure rates were 85%. This procedure ushered in a minimally invasive surgical approach for treating patients with SUI. The retropubic (RP) sling has been widely accepted as the gold standard for the treatment of SUI (stress urinary incontinence)."

Dr. Iakovlev's review article also incorporates the following statement: "In comparison with patients with an RP sling, patients with a TOT sling have a higher incidence of persistent pain (32% versus 10%) and dyspareunia (18% versus 3%)." (Int. Urogynecol. J. 23, 321–325, 2012) is disingenuous as the authors of this article state: "we were unable to assess the incidence of complications due to lack of a denominator." In other words, Dr. Iakovlev did not note that this study looked *only* at patients who had complications, as opposed to the overall number of women who received the implant. Significantly, Dr. Iakovlev's general report in these Wave 1

cases does not compare the incidences of pain or other complications with other surgical approaches to pelvic relaxation without the use of implanted materials.

Dr. Iakovlev theorizes a link between the presence of inflammation adjacent to nerve "receptors" and pain, but he does not propose an actual mechanism. Nor does he – or is able to – identify a specific type of receptor to which he refers, if they can be documented by histology, or how a nerve branch interrupted by surgical implantation would even be capable of supporting a functional receptor of any type. Likewise, Dr. Iakovlev's report provides no examples of receptors he has identified.

Dr. Iakovlev also states, " . . . [i]nflammatory mediators causing hypersensitivity of pain and other (touch etc.) receptor. This leads to pain to touch or with movement, and if a stimulus is sufficiently high, it can cause pain at rest." He does not specify the specific inflammatory mediators to which he refers among the many listed in this reference. Furthermore, there is no foundation for even invoking the inflammatory processes described in this reference given the inflammation noted microscopically in mesh explants. As discussed below, Dr. Iakovlev provides no explanation why fewer than 5% of patients undergoing explanation of SUI slings wholly or in part for pain experience this pain due to inflammation when 100% of mesh implants are associated with a normal inflammatory response to biosynthetic materials. Nor does he explain why de novo dyspareunia would be comparable between pelvic organ prolapse repair with mesh and native tissue repair, the latter of which does not involve the implantation of a biosynthetic material and its associated normal inflammatory response.

Dr. Iakovlev's pattern of scientifically unreliable assumptions continues with his description of the scarring process in periurethral folds that results in dyspareunia in some individuals who had undergone transobturator sling placement. For instance, Dr. Iakovlev states,

"Unfortunately, no data were presented describing the histopathology of excised tissue that was removed owing to painful banding, although little imagination is required to understand how this effect could cause pain (Figure 9)". But Figure 9 consists of a gross photograph of explanted sling materials with no mention if the patient experienced pain. Dr. Iakovlev's method consists of offering imaginary concepts as a form of proof. But while imagination plays an important role in scientific discovery, hypotheses are only valid to the extent they are proven using an accepted scientific methodology.

Tellingly, Dr. Iakovlev acknowledges in his review article that "we have an incomplete understanding of interactions specific to a mesh material and design as well as the pathophysiology of any complications." Dr. Iakovlev's opinions regarding the incidental finding of nerve twigs adjacent to mesh explants and mechanisms of pain illustrate his "incomplete understanding."

**Response to Dr. Iakovlev's Expert Report**

Dr. Iakovlev's expert report recapitulates the erroneous link between fibrosis and/or inflammation with pain in a small percentage of patients having undergone mesh explantation, as described above. Dr. Iakovlev describes "ingrowth" of nerves as a cause of pain and depicts nerve twigs near mesh in photomicrographs but offers no actual pathological mechanism whereby nerve twigs adjacent to mesh equate with pain. These nerve twigs are histologically normal aside from the artifact of formalin fixation and paraffin embedding described above, and they show no features of traumatic neuromas. The only information this finding provides is that the vaginal wall and surrounding tissue contain nerve fibers that can send various signals. These findings provide no evidence of what signals the fibers may be capable of carrying, if any, where their receptors are located, or where they connect with the CNS.

In addition, Dr. Iakovlev also describes a novel compartment syndrome associated with mesh tissue incorporation, which is unprecedented in the surgical or pathology medical literature regarding synthetic sling surgery. Dr. Iakovlev invokes his hypotheses about the significance of nerve regeneration in this context. Again, he does not identify a mechanism beyond pure conjecture, or identify actual "receptors" or a substance derived from inflammation that might contribute to the hypothetical pain related to nerve regeneration in the ingrown "scar" or near folded implants. Importantly, Dr. Iakovlev has recently testified that he does not know whether folding or curling of mesh occurred at the time of placement in the body. (Sept. 11, 2015 Deposition of Dr. Iakovlev, p. 215) (stress urinary incontinence case); (Aug. 12, 2014 Deposition of Dr. Iakovlev, p. 155) (pelvic organ prolapse case). Thus, his theory that the properties of the mesh lead to folding or curling that result in pain is unreliable.

The most problematic deficiency in the assertions by Dr. Iakovlev is the complete lack of foundation for the claim that the presence of nerve twigs and inflammation, as well as any other histological finding, is causally related to pain. The scientific method would require that meshes removed for reasons other than pain be examined for the presence of nerve twigs and other findings and compared with those removed from patients with pain. Dr. Iakovlev does not exclude the likelihood that that the same findings of nerve twigs, inflammation, hypothetical features of compartmentalization, etc. would be found in patients without pain, dyspareunia or other neurological symptoms. Dr. Iakovlev's recent article in Hernia did not control for foreign body reaction, inflammation, fibrosis, degree of "deformation", etc., so it fails to provide any foundation for his current opinions.

Stated differently, Dr. Iakovlev has not provided any criteria of any sort whereby an independent and unbiased microscopist could make a link between the presence of incidental

nerve twigs and the usual inflammation associated with implanted mesh, and pain symptoms in a small minority of patients reporting pain versus the significant majority of symptom-free patients having undergone synthetic sling implantation. The same can be said for Dr. Iakovlev's assertions regarding fibrosis, its effects on nerves, and any link to pain.

I have the following overall comments about the figures in Dr. Iakovlev's report: None of the illustrations demonstrates the pathologic features of traumatic neuroma. None of these illustrations shows any nerve receptors, despite Dr. Iakovlev's theories regarding the involvement of hypothetical nerve receptors in mediating alleged pain originating in nerves adjacent to mesh. Observation of nerve twigs in tissue is not the same as identification and typing of sensory receptors. All of the nerves depicted by hematoxylin and eosin (H&E) or immunohistochemical studies (neurofilament and S100) appear unremarkable, notwithstanding the usual artifacts of formalin fixation, paraffin embedding, and displacement by the mesh in a subset of nerve fibers. Mere presence in fibrous connective tissue is not reliable evidence of any pathology for any given nerve. The mere presence of nerve twigs in tissue is not a reliable indicator of sensation. The presence of neural ganglia is of no significance other than to demonstrate the innervation attributable to ganglia is of an autonomic, not pain transmitting, nature. The ganglia shown in Dr. Iakovlev's report show no signs of pathologic change.

In addition, I have the following specific opinions about Dr. Iakovlev's figures:

1) Figure sets 1a-c depict the normal and expected foreign body response to implanted material. Dr. Iakovlev himself testified that this is a non-specific reaction to a foreign body. (Sept. 11, 2015 Deposition Transcript of Dr. Iakovlev at pp. 93-94. Note that these figures are the same as his Mullins report).

2) Figure 2a shows that normal tissue, by Dr. Iakovlev's account, is present remarkably near mesh material, estimated to be less than 100 microns (less than 1/10$^{th}$ of a millimeter) from the mesh. (Sept. 11, 2015 Deposition Transcript of Dr. Iakovlev at pp. 112-113). The photos in figures 2b also depicts fibrosis and inflammation limited to a very small area immediately surrounding mesh.

3) Figure set 2g shows putative smooth muscle identified by smooth muscle actin immunohistochemistry. The significance Dr. Iakovlev attaches to the presence of smooth muscle is stated in his deposition of September 11, 2015, whereby he considers this evidence of migration of implanted mesh to a region with native vaginal or periurethral smooth muscle. In fact, smooth muscle is not only capable of proliferation in the vicinity of a surgical wound, but it is an important part of normal healing. ("Inflammation and repair", Chapter 3, In: Robbins and Cotran. Pathologic Basis of Disease (9$^{th}$ edition)).

4) Figure 3a shows nerve twigs adjacent to mesh in fibrous tissue. By H&E stain, these nerve twigs show no pathologic alteration.

5) Figure 3b and 3c – It is subjective to say these nerves are severely distorted. There is no evidence of any clinical consequence. The photos show very little inflammation and minimal foreign body response.

6) Figure 3d – The figure is incorrectly labeled as being stained with H&E, which it is not. As a result, the exact stain is unknown. The photos in the upper panel could be the result of any surgical incision. The legend describing the lower panel incorrectly asserts that "separation of fascicles in the scar tissue = traumatic neuroma". A traumatic neuroma would show signs of axonal sprouting, which is not shown in

these photos. There is insufficient evidence to label the lower panel as a traumatic neuroma. Traumatic neuromas usually produce mass effect ("-oma" denotes tumors and other abnormal growths, therefore "neuroma" signifies a focal enlargement of nerve), contain schwannosis, fibrosis, and the aforementioned axonal sprouting.

7) Figure 3e – There is no evidence of axonal sprouting necessary to label this a traumatic neuroma.

8) Figure 3f – The top panel in this figure shows a nerve displaced by mesh with no evidence of clinical consequences.

9) Figure 3g – This figure shows nerves displaced by mesh. Rather than attribute pathology, it is noteworthy that the nerve is intact despite its proximity to the mesh fiber. Again, the separation of fascicles is not the definition of a traumatic neuroma. The ganglion in the top panel shows no abnormal findings.

10) Figure 3h -- shows unremarkable nerves in which some S100 positive small structures may not be nerve fibers. There is nothing concerning about these nerves or the tissue in which they are located.

11) Figure 3g -- depicts nerve in in the middle of the mesh pore and it appears normal with no evidence of injury. There is nothing concerning about these nerves or the tissue in which they are located.

12) Figures 3h -- The "degeneration" of the nerve is actually not degeneration at all. The feature shown is most likely a Renaut body and, despite Dr. Iakovlev's figure legend, Renaut bodies are a normal finding in peripheral nerves that may not be recognized by a surgical pathologist inexperienced in nerve pathology (Midroni G and Bilbao JM. "Biopsy Diagnosis of Peripheral Neuropathy" 1995, Ch 2, pp. 27-29). In any

event, Dr. Iakovlev testified that degeneration of nerves would not be painful, but would result in numbness. (Sept. 11, 2015 Deposition Transcript of Dr. Iakovlev at p. 159).

13) Figure set 4a – These show normally appearing nerves and ganglia with no pathology depicted.

14) Figure set 4b, 4c, 4d, and 4e – This figure shows a normally appearing neural ganglion. Dr. Iakovlev's assertion that the ganglion is "affected" is unclear, as the ganglion shows no pathologic change. Noteworthy about this ganglion is the perfect appearance less than millimeter from the mesh fiber.

15) Figure set 5 -- demonstrates S100 staining pattern identifying submucosal nerve twigs and some nonspecific epidermal staining. Dr. Iakovlev uses this figure to extrapolate that there are nerves between hardened/stiffened mesh and the vaginal mucosa, a situation that he asserts would be painful. Dr. Iakovlev has no reliable scientific basis for this opinion.

16) Figure set 6a, 6b, and 6c -- These figures do not provide sufficient evidence to diagnose edema. Edema is most reliably determined by gross examination of tissues by surgeons or pathologists and is ideally confirmed microscopically when such a gross determination has been made. Nonetheless, edema is a common feature of hundreds of pathological processes which are not associated with pain. Dr. Iakovlev does not provide any evidence or explanation as to why edema associated with mesh implants may be associated with pain and why this hypothetical edema causes pain in a minority of patients and not the vast majority of mesh implant recipients.

17) Figure 7a and 7b – these display normal appearance after any surgery.

18) Figure set 7c – the importance of this photo is unknown as there is not mesh in the field.

19) Figure set 7d, 7e, 7f and 7g – these images show the expected appearance after surgery in any field containing muscle and nerve twigs. It is unlikely that the muscle shown in these photos has much, if any function, due to the scattered single clusters.

20) Figure set 9a – it is not clear how Dr. Iakovlev reached his conclusions regarding obliterated vessels as the figure does not demonstrate evidence that the changes were due to the mesh and any such changes to vessels can occur with surgery alone.

21) Figure 9b shows possible thrombosis, commonly found in operative sites and of doubtful clinical significance.

I have recently received and will be reviewing the histology slides for several specimens from patients in this grouping of cases. My findings on those cases will be contained in separate reports for each plaintiff.

**Conclusion**

Dr. Iakovlev asserts that incidentally found nerve twigs in pathology slides demonstrate that a patient suffers pain as a result of mesh implantation. The methodology used by Dr. Iakovlev is not accepted by the scientific community because he does not use the scientific method to reach his conclusions. In addition to ignoring scientific literature that contradicts his opinion, Dr. Iakovlev fails to compare his findings to a comparator group, and reaches a number of conclusions that are contrary to the generally accepted principles of neuropathology. There is nothing in Dr. Iakovlev's assertions to allow a correlation through any demonstrable pathological mechanism between incidental nerve twigs and pain. Additionally, Dr. Iakovlev fails to account for the fact that the vast majority of patients implanted with Ethicon's TVT meshes and most

patients implanted with Ethicon's pelvic organ prolapse meshes do not undergo removal with pain as a complaint. Furthermore, Dr. Iakovlev's opinions are not supported by the histology, which shows the normal healing process in the vicinity of an implanted mesh.

Dr. Iakovlev bases his opinions upon erroneous assumptions which he offers despite his lack of subspecialty training or expertise in the pathology of the peripheral nervous system. These opinions do not carry any significance whatsoever in explaining pain that was allegedly experienced by these plaintiffs as a result of their repair surgery and implanted mesh.

**Compensation**

My hourly rate for work in this case is $500.

DATE: 3/1/2016

HANNES VOGEL, M.D.

CURRICULUM VITAE

# HANNES VOGEL, MD

**HOME ADDRESS:**

[redacted]

**BUSINESS ADDRESS:**

[redacted]

**EDUCATION:**

| | |
|---|---|
| 1975 | BA (Biology), Rice University, Houston, TX |
| 1980 | MD, Baylor College of Medicine, Houston, TX |

**INTERNSHIPS AND RESIDENCIES:**

| | |
|---|---|
| 1980-82 | Resident in Pediatrics, Baylor Affiliated Hospitals, Houston, TX |
| 1982-83 | Resident in Pediatrics, University of California, San Francisco, CA |
| 1983-84 | Chief Resident, Pediatrics, University of California, San Francisco, CA |
| 1984-87 | Resident in Anatomic Pathology, Beth Israel Hospital/Harvard Medical School, Boston, MA |
| 1987-89 | Resident in Neuropathology, Stanford University Hospital/Stanford University School of Medicine, Stanford, CA |
| 2000-2001 | Resident in Pediatric Pathology, Texas Children's Hospital, Houston, TX |

**LICENSURE AND CERTIFICATIONS:**

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| 1980 | Texas License Registration No. F7269. Expiration 8-31-2002 |
| 1982 | California License Registration No. C040662. Expiration 5-31-2014 |
| 1986 | American Board of Pediatrics |
| 1989 | American Board of Pathology: Combined Anatomic and Neuropathology<br>Board Eligible: Pediatric Pathology |

**PROFESSIONAL MEMBERSHIPS:**

United States and Canadian Academy of Pathology
American Association of Neuropathologists
Associate, American Academy of Neurology
German Association of Neuropathology and Neuroanatomy
California Society of Pathologists
South Bay Pathology Society
Baltic Association of Neuropathology

**ACADEMIC APPOINTMENTS:**

| | |
|---|---|
| 1985-87 | Affiliate Staff Member, Department of Pediatrics, Carney Hospital/Tufts University School of Medicine, Boston, MA |
| 1988-90 | Clinical Associate, Department of Pediatrics, University of California, San Francisco, CA |
| 1989-90 | Physician Specialist and Clinical Instructor, Department of Pathology, Stanford University Hospital, Stanford, CA |
| 1991-92 | Assistant Professor, Departments of Medical and Surgical Neurology, and Pathology, and Pediatrics, Texas Tech University - Health Sciences Center, Lubbock, TX |
| 1992-99 | Assistant Professor, Departments of Pathology and Pediatrics, Baylor College of Medicine, Houston, TX |
| 1997-2002 | Assistant Professor, Department of Pathology and Laboratory Medicine, Texas A&M University Health Science Center, Houston, TX |

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| 1999-2002 | Associate Professor, Departments of Pathology and Pediatrics, Baylor College of Medicine, Houston, TX |
| 2001-2002 | Director of Neuropathology, Texas Children's Hospital, Houston, TX |
| 5/8/02 – 5/31/02 | Acting Associate Professor of Pathology, Stanford University School of Medicine, Stanford, CA |
| 6/1/02 – 4/30/07 | Associate Professor of Pathology and Pediatrics (Medical Genetics) Stanford University School of Medicine, Stanford, CA |
| 6/1/02 – | Director of Neuropathology, Stanford University Medical Center, Stanford, CA |
| 2005- | Associate Chair of Neuropathology, as Member of the Department of Pathology Leadership Group, Stanford University, Stanford, CA |
| 9/1/05- 4/30/07 | Associate Professor of Neurosurgery (by courtesy), Stanford University School of Medicine, Stanford, CA |
| 5/1/07- | Professor of Pathology and Pediatrics (Medical Genetics) and Neurosurgery (by courtesy), Stanford University School of Medicine, Stanford, CA |
| 2008- | Member, Stanford Cancer Center, Stanford University Hospital, Stanford, CA |

**EDITORIAL BOARDS:**

Executive Advisory Board, Clinical Neuropathology
Czeho-Slovak Pathology and Forensic Medicine

**AD HOC REVIEW:**

Acta Neuropathologica
Archives of Pathology and Laboratory Medicine
Brain Pathology
Human Pathology
Journal of Neuropathology and Experimental Neurology
Plos One

**AWARDS AND HONORS:**

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| June 11, 2005 | Henry J. Kaiser Family Foundation Award for Excellence in Preclinical Teaching |

**MAJOR COMMITTEE ASSIGNMENTS:**

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| 2003-2006 | Awards Committee, American Association of Neuropathologists |
| 2005- | American Academy of Neurology Residency Inservice Training Examination (RITE) Committee, Neuropathology section |
| 2002-2006 | Alternate member, Stanford University Institutional Review Board |
| 2006-2007 | Voting member, Stanford University Institutional Review Board |
| 2006-2009 | Alternate member, Stanford School of Medicine Faculty Senate |
| 2006-2007 | Admissions Committee, Stanford University School of Medicine |
| Oct 2-4, 2013 | Co-Chair, Fundamentals of Cancer Biology<br>Banbury Conference Center, Cold Springs Harbor Laboratory |

**RESEARCH GRANTS:**

*Past:*

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| 1988-90 | Collaborative research with Drs. Eugene Butcher and Louis Picker, Stanford University Medical School and Palo Alto Veteran's Administration Hospital: Expression of CD44 (HCAM) by human astrocytes, supported by American Heart Association, NIH grant AI19957, and an award from the Weingart Foundation. |
| 1992-1993 | Role of CD44 in astrocyte neoplasia, immunolocalization of CD44 in the central and peripheral nervous systems, CD44 as a astrocytic hyaluronate receptor. Supported by Baylor College of Medicine, Department of Pathology institutional grants, and the Moran Foundation. |
| 1992-1994 | Collaboration with Drs. Phillip Soriano and Paul Stein: characterization of renal pathology in transgenic mice homozygous for deletion of tyrosine kinases fyn and yes, supported by the Howard Hughes Medical Institute. |
| 1992-1994 | Pilot project investigator in the role of astrocytic adhesion molecules in Alzheimer's disease. Alzheimer Disease Research Center, Baylor College of Medicine. NIH-AG08664. |

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| 1993-1995 | Collaboration with Lawrence Donehower: characterization of neoplasia arising in p53- and Rb-deficient mice, supported by National Cancer Institute CA 54897. |
| 1993-1997 | Collaboration with Mark Perin: Immunohistochemical analysis of novel synaptic proteins, supported by National Institutes of Health. RO1 NS-30541. |
| 1993-1997 | Collaboration with Dr. Donald M. Marcus, Baylor College of Medicine, in the histopathological analysis of rat tissues in the study of an animal model of autoimmune neuropathy. |
| 1995-1997 | Collaboration with Dr. Russell Lebovitz, Department of Pathology, Baylor College of Medicine, in the analysis of the neuropathological abnormalities in mice deficient in superoxide dismutase 2. |
| 1995-2002 | Collaboration with Dr. Allan Bradley, Department of Molecular Genetics, in the analysis of tumorigenesis in transgenic mice lacking p53, mdm, blm. |
| 1998-2003 | Pathologist, NIH Program Project: Genetic Analysis of Mouse Chromosome 11. PI: Allan Bradley, Ph.D. |
| | Co-investigator, Pediatric Brain Tumor Clinical Trials Consortium. Granting agency: National Cancer Institute. |
| 1999-2002 | Consultant pathologist, Lexicon Genetics Inc. |
| 1999-2002 | Pathologist: “Inactivated p53 Gene in Mice” R01 CA54897-07/10 09/30/99 - 07/31/03. |
| 2000-2002 | New Investigator, Center for AIDS Research, Baylor College of Medicine. |
| 2005-2007 | Principal Investigator – Immunohistochemistry of mitochondrial respiratory chain complexes. Pediatric Health Research Fund Award. |

*Present:*

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| 2002- | Collaborator – Role of p63 in development and neoplasia. PI Alea Mills, Cold Spring Harbor Laboratory, NY. |
| 2002- | Collaborator – Neural Stem Cell Research. PI Theo Palmer, Department of Neurosurgery, Stanford University. |

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| 2002- | Collaborator – Mouse model of spongiform degeneration. PI Greg Barsh, Stanford University. |
| 2004- | Collaborator – Role of defective chromosome repair in neoplasia. PI Guangbin Luo, Case Western University. |
| 2004- | Collaborator – Parkinson disease-related genes in genetically engineered drosophila. PI Bingwei Lu, Stanford University. |
| 2004- | Collaborator – Role of gigaxonin and other cytoskeletal proteins in axonal function. PI Yanmin Yang, Stanford University. |
| 2005- | Collaborator – Role of chromosome 1p in neoplasia, PI Alea Mills, Cold Spring Harbor Laboratory, NY. |
| 2006- | Collaborator – Role of Ku80 in medulloblastoma and senescence. PI Paul, D.V.M., Department of Molecular Medicine, University of Texas Health Science Center at San Antonio. |
| 2007- | Other Investigator – Pathophysiology of lysosomal free sialic acid storage disorders. NIH. |
| 2007- | Other Investigator – Immunology of neural stem cell fate and function. California Institute of Regenerative Medicine. |
| 2008- | Collaborator – Role of Beta-receptor signaling in cardiomyopathy. PI Daniel Bernstein, Cardiovascular Institute, Stanford School of Medicine. |
| 2008- | Principal Investigator – Genetic dissection of mitochondria and muscle maintenance. Palo Alto Institute for Research Education. |
| 2008- | Collaborator – Prognostic value of MRI and biomarker in comatose post-cardiac arrest patients. PI Christine Wijman, Stanford Stroke Center, Stanford School of Medicine. |

**TEACHING EXPERIENCE:**

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| 1983-84 | Organization and presentation of clinical conferences, ward rounds, and grand rounds in general pediatrics, San Francisco General Hospital. |
| 1985-87 | Laboratory instructor general pathology, Harvard Medical School. |
| 1987-90 | Lecturer in medical school core curriculum neuropathology lectures; organization and presentation of monthly surgical and autopsy neuropathology conference, Department of Pathology, Stanford University School of Medicine. |

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| 1991-92 | Organized comprehensive neuropathology training for neurology residents and core neuropathology curriculum Texas Tech University - Health Sciences Center medical students. |
| 1992- | Lecturer in general and neuropathology for Baylor College of Medicine students, pathology residents, neurology residents, neuropathology fellows, neurosurgical conferences, monthly pediatric nerve and muscle pathology conference. |
| 1994 | Invited speaker First Annual J. Edward Stern, M.D. Neuroscience Symposium, Providence Memorial Hospital, El Paso, Texas. "Neuropathology of common neurological disorders: An update". |
| 1995 | Invited speaker, Annual meeting of the Texas Society for Histotechnology, Inc., Houston Texas. "Current concepts in processing nerve and muscle biopsies". Half-day Wet Workshop. April 9. |
| 1995-2001 | Invited lecturer. Topics in neuropathology, core curriculum in pathology. Texas A&M School of Medicine. |
| 1995-1999 | Invited lecturer. Topics in neuropathology. Texas Tech University School of Medicine. |
| 2001 | Faculty, After Dinner Seminar: “Pediatric Neuropathology”. Annual meeting, American Academy of Neurology, Philadelphia, PA, May 7. |
| | Pathology department representative, selected to lecture to entering medical school class, Core Concepts Curriculum, Baylor College of Medicine. |
| 2002- | Lecturer in neuropathology and pediatric neoplasia, Stanford University School of Medicine. |

**BIBLIOGRAPHY:**

*Peer-Reviewed Articles:*

1. Eidelberg D, Sotrel A, **Vogel H**, Walker P, Kleefield J & Crumpacker CS, 3rd. Progressive polyradiculopathy in acquired immune deficiency syndrome. *Neurology* 36, 912-916, (1986). PMID 3012412.

2. Schnitt SJ & **Vogel H**. Meningiomas. Diagnostic value of immunoperoxidase staining for epithelial membrane antigen. *Am J Surg Pathol* 10, 640-649, (1986). PMID 2428264.

3. Shiurba RA, Eng LF, **Vogel H**, Lee YL, Horoupian DS & Urich H. Epidermal growth factor receptor in meningiomas is expressed predominantly on endothelial cells. *Cancer* 62, 2139-2144, (1988). PMID 3052782.

4. **Vogel H**, Horoupian DS & Silverberg G. Do folliculo-stellate adenomas of the pituitary gland exist or are they intrasellar meningiomas? *Acta Neuropathol* 77, 219-223, (1988). PMID 2465659.

5. **Vogel H**, Halpert D & Horoupian DS. Hypoplasia of posterior spinal roots and dorsal spinal tracts with arthrogryposis multiplex congenita. *Acta Neuropathol* 79, 692-696, (1990). PMID 2360413.

6. **Vogel H**, Gessaga EC, Horoupian DS & Urich H. Aqueductal atresia as a feature of arhinencephalic syndromes. *Clin Neuropathol* 9, 191-195, (1990). PMID 2225595.

7. **Vogel H**, Urich H, Horoupian DS & Wertelecki W. The brain in the 18q-syndrome. *Dev Med Child Neurol* 32, 732-737, (1990). PMID 2210088.

8. Raisanen J, **Vogel H** & Horoupian DS. Primitive pineal tumor with retinoblastomatous and retinal/ciliary epithelial differentiation: an immunohistochemical study. *J Neurooncol* 9, 165-170, (1990). PMID 2262801.

9. Honig LS, Snipes GJ, **Vogel H** & Horoupian DS. Sensorimotor neuropathy in hemophagocytosis syndrome. *Acta Neurol Scand* 84, 316-320, (1991). PMID 1663307.

10. Page KA, **Vogel H** & Horoupian DS. Intracerebral (parenchymal) infusion of methotrexate: report of a case. *J Neurooncol* 12, 181-186, (1992). PMID 1560265.

11. **Vogel H**, Butcher EC & Picker LJ. H-CAM expression in the human nervous system: evidence for a role in diverse glial interactions. *J Neurocytol* 21, 363-373, (1992). PMID 1607880.

12. **Vogel H** & Horoupian DS. Filamentous degeneration of neurons. A possible feature of cytosine arabinoside neurotoxicity. *Cancer* 71, 1303-1308, (1993). PMID 8435808.

13. Stein PL, **Vogel H** & Soriano P. Combined deficiencies of Src, Fyn, and Yes tyrosine kinases in mutant mice. *Genes Dev* 8, 1999-2007, (1994). PMID 7958873.

14. Galasso PJ, Stanton MS & **Vogel H**. Propafenone-induced peripheral neuropathy. *Mayo Clin Proc* 70, 469-472, (1995). PMID 7731257.

15. Schlimgen AK, Helms JA, **Vogel H** & Perin MS. Neuronal pentraxin, a secreted protein with homology to acute phase proteins of the immune system. *Neuron* 14, 519-526, (1995). PMID 7695898.

16. Tan TQ, **Vogel H,** Tharp BR, Carrol CL & Kaplan SL. Presumed central nervous system Whipple's disease in a child: case report. *Clin Infect Dis* 20, 883-889, (1995). PMID 7540874.

17. Matzuk MM, Lu N, **Vogel H**, Sellheyer K, Roop DR & Bradley A. Multiple defects and perinatal death in mice deficient in follistatin. *Nature* 374, 360-363, (1995). PMID 7885475.

18. Harvey M, Vogel H, Lee EY, Bradley A & Donehower LA. Mice deficient in both p53 and Rb develop tumors primarily of endocrine origin. *Cancer Res* 55, 1146-1151, (1995). PMID 7867001.

19. Harvey M, **Vogel H**, Morris D, Bradley A, Bernstein A & Donehower LA. A mutant p53 transgene accelerates tumour development in heterozygous but not nullizygous p53-deficient mice. *Nat Genet* 9, 305-311, (1995). PMID 7773294.

20. Rowen JL, Doerr CA, **Vogel H** & Baker CJ. Balamuthia mandrillaris: a newly recognized agent for amebic meningoencephalitis. *Pediatr Infect Dis J* 14, 705-710, (1995). PMID 8532431.

21. Donehower LA, Harvey M, **Vogel H**, McArthur MJ, Montgomery CA, Jr., Park SH, Thompson T, Ford RJ & Bradley A. Effects of genetic background on tumorigenesis in p53-deficient mice. *Mol Carcinog* 14, 16-22, (1995). PMID 7546219.

22. Lebovitz RM, Zhang H, **Vogel H**, Cartwright J, Jr., Dionne L, Lu N, Huang S & Matzuk MM. Neurodegeneration, myocardial injury, and perinatal death in mitochondrial superoxide dismutase-deficient mice. *Proc Natl Acad Sci U S A* 93, 9782-9787, (1996). PMID 8790408. PMCID 38506.

23. Jones SN, Sands AT, Hancock AR, **Vogel H**, Donehower LA, Linke SP, Wahl GM & Bradley A. The tumorigenic potential and cell growth characteristics of p53-deficient cells are equivalent in the presence or absence of Mdm2. *Proc Natl Acad Sci U S A* 93, 14106-14111, (1996). PMID 8943068. PMCID 19502.

24. Bhattacharjee MB, Armstrong DD, **Vogel H** & Cooley LD. Cytogenetic analysis of 120 primary pediatric brain tumors and literature review. *Cancer Genet Cytogenet* 97, 39-53, (1997). PMID 9242217.

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29. Venkatachalam S, Shi YP, Jones SN, **Vogel H**, Bradley A, Pinkel D & Donehower LA. Retention of wild-type p53 in tumors from p53 heterozygous mice: reduction of p53 dosage can promote cancer formation. *EMBO J* 17, 4657-4667, (1998). PMID 9707425. PMCID 1170795.

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32. Mills AA, Zheng B, Wang XJ, **Vogel H**, Roop DR & Bradley A. p63 is a p53 homologue required for limb and epidermal morphogenesis. *Nature* 398, 708-713, (1999). PMID 10227293.

33. **Vogel H**, Lim DS, Karsenty G, Finegold M & Hasty P. Deletion of Ku86 causes early onset of senescence in mice. *Proc Natl Acad Sci U S A* 96, 10770-10775, (1999). PMID 10485901. PMCID 17958.

34. **Vogel H.** Pathologic findings in nerve and muscle biopsies from 47 women with silicone breast implants. *Neurology* 53, 293-297, (1999). PMID 10430416.

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57. Scaglia F, Towbin JA, Craigen WJ, Belmont JW, Smith EO, Neish SR, Ware SM, Hunter JV, Fernbach SD, Vladutiu GD, Wong LJ & **Vogel H**. Clinical spectrum, morbidity, and mortality in 113 pediatric patients with mitochondrial disease. *Pediatrics* 114, 925-931, (2004). PMID 15466086.

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62. Massengale M, Wagers AJ, **Vogel H** & Weissman IL. Hematopoietic cells maintain hematopoietic fates upon entering the brain. *J Exp Med* 201, 1579-1589, (2005). PMID 15897275. PMCID 2212913.

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73. Adam MP, Chueh J, El-Sayed YY, Stenzel A, **Vogel H**, Weaver DD & Hoyme HE. Vascular-type disruptive defects in fetuses with homozygous alpha-thalassemia: report of two cases and review of the literature. *Prenat Diagn* 25, 1088-1096, (2005). PMID 16231329.

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# Hannes Vogel

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| **Company Witnesses** |
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| Barbolt, Thomas [1.8.14] |

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**Expert Reports**
Iakovlev, Vladimir (General) - 1.29.16

**Trials and Depositions**
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